Evaluation of Thoracic Injury in Swine Model with a Noise Immune Stethoscope

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14. ABSTRACT

Auscultation of heart and lung sounds is an important component of casualty triage, and military health care providers rely on this in order to manage an airway and to provide life saving interventions. This study evaluated the feasibility and sensitivity of a newly developed electronic stethoscope concept based on both enhanced acoustic isolation technology and on the application of a Doppler sensor (2 to 3 MHz) insensitive to noise audible to the human ear. The study used seven female Yorkshire swine. In each experiment, the noise immune stethoscope was used in both acoustic and Doppler modes to auscultate heart and lung sounds. Findings with the acoustic mode were more consistent with traditional auscultation when the endotracheal tube was placed within the esophagus and when a pnuemothorax was induced. In contrast, definitive Doppler findings were difficult to elicit and appeared neither sensitive to nor specific of the induced pathology. Overall, the acoustic mode performed well in this study while the Doppler mode was limited in its current diagnostic format.

15. SUBJECT TERMS

noise immune stethoscope (NIS), acoustic, Doppler, Yorkshire swine, pneumothorax, white noise, flight surgeon, auscultation, chest tube, needle decompression

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Introduction

Auscultation of heart and lung sounds is an important component of casualty triage and ongoing care; it is a tool used by military health care providers to identify pathophysiology and determine the appropriate course of treatment.

Field assessment and aeromedical evacuation of wounded Soldiers present unique challenges for patient care; the most notable is the lack of effective auscultation for lung sounds to assess airway patency and adequate ventilation. Whilst devices have been developed to measure end-tidal carbon dioxide (CO_2) as an indicator of endotracheal tube placement, more direct and familiar methods are desired for field use and during transportation. The inability of medics or flight surgeons to hear heart and lung sounds or detect the development, or progression, of a pneumothorax or endotracheal tube displacement greatly compromises their ability to manage the airway and provide appropriate life saving interventions.

The standard acoustic stethoscope operates on the transmission of sound from the chest-piece, via air-filled hollow tubes, to the listener's ears. The chest-piece usually consists of two sides that can be placed against the patient for sensing sound: a diaphragm (plastic disc) or bell (hollow cup). If the diaphragm is placed on the patient, body sounds vibrate the diaphragm, creating acoustic pressure waves which travel up the tubing to the listener's ears. If the bell is placed on the patient, the vibrations of the skin generate acoustic pressure waves which travel up to the listener's ears. The bell transmits low frequency sounds, while the diaphragm transmits higher frequency sounds. One problem with the acoustic stethoscope is that the sound level is extremely low, making diagnosis difficult in the presence of any significant environmental sound.

This report presents data evaluating the feasibility and sensitivity of a newly-developed, electronic, noise immune stethoscope (NIS) concept based on both enhanced acoustic isolation technology and on the application of a Doppler sensor (2 to 3 megaHertz [MHz]) insensitive to noise audible to the human ear, instead detecting tissue movement and blood flow. The device used was an advanced prototype as developed by Active Signal Technologies (AST), Inc of Linthicum Heights, Maryland, in conjunction with the U. S. Army Aeromedical Research Laboratory (USAARL). The prototype was the most recent model available in September 2008.

Methods

Experiments were performed using seven[1] female Yorkshire swine (45 to 50 kilograms [kg]). All procedures were reviewed and approved by the animal care and use review office of the U. S. Army Medical Research and Materiel Command (USAMRMC) and the U. S. Army Institute of Surgical Research (USAISR), Brooke Army Medical Center (BAMC) Animal Care and Use Committee.

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[1] Eight animals were entered into the study. One animal died from cardiac tamponade shortly after instrumentation and thus, only seven animals were analyzed.

Surgical preparation

Seven animals were anesthetized with an intramuscular injection of Telazol (tiletamine HCl/zolazepam HCl) at 4 to 6 milligrams (mg)/kg and prior to intubation they were pre-medicated with glycopyrrolate at 0.004 to 0.01 mg/kg intramuscular (IM) to reduce the vagal reflex on intubation and reduce airway secretions. Animals were intubated with an endotracheal tube (7 millimeters [mm] by 55 centimeters [cm]) and anesthesia was maintained with 1 to 5% isoflurane with 100% oxygen (O_2) throughout the instrumentation period. Core temperature was maintained at 37 to 39 degrees Celsius (°C).

Catheterization

A fluid-filled Tygon catheter was inserted into the left carotid artery for continual pressure (systolic, diastolic, and mean) and heart rate monitoring. An 8.0 French (Fr) catheter introducer was placed in the right jugular vein. A continuous Swan-Ganz catheter threaded through the 8.0 Fr introducer provided continuous cardiac output, central venous pressure, and pulse oximeter oxygen saturation (SpO_2). All continuous physiologic signal data were collected using the USAISR-developed data acquisition system (DAQ). A catheter with a three-way stopcock was placed and secured into a marginal ear vein of the pig for administration of intravenous (IV) drugs. Once IV access was established, the isoflurane was turned off and anesthesia was maintained with midazolam (12.5 mg/milliliter [ml]) 0.1 to 0.5 mg/kg IV bolus followed by an IV continuous rate infusion (CRI) started at 1.5 mg/kg/hour (hr) and a bolus of ketamine at 33 mg/kg (100 mg/ ml) followed by a CRI at 10 to 33mg/kg/hr. The rates of each drug were adjusted as needed to affect a surgical plane of anesthesia. To ensure a proper degree of analgesia, fentanyl CRI at 0.03 to 0.1 mg/kg/hr was administered as needed. Ventilation was supported using a Hallowell ventilator and medical air (21% O_2). The combination of ketamine and midazolam was used to minimize cardio-depressive anesthetic effects.

Procedures

In each experiment, the NIS was used in both acoustic and Doppler modes to auscultate heart and lungs sounds for each experiment. All auscultation ratings were made by the same experienced investigator/physician. The recording positions (figure 1) were marked on each animal to minimize variation of subsequent recordings within a particular animal and across all other animals. These recording sites consisted of three lung sound locations (apex, mid, and base) on the left and right chest, respectively, and one heart sound site located in the left para-sternal region.

Firstly, in the control experiment, the endotracheal tube (ET) was correctly placed in the trachea. Heart and lungs sounds were recorded in the intact chest. The ET tube was then placed incorrectly into the esophagus, alongside a gastric tube (to prevent excessive gas build-up), and the recording sequence repeated. The ET tube was then replaced in the trachea for the remainder of the experiments. After a 10-minute recovery period, experiment 2 was initiated.
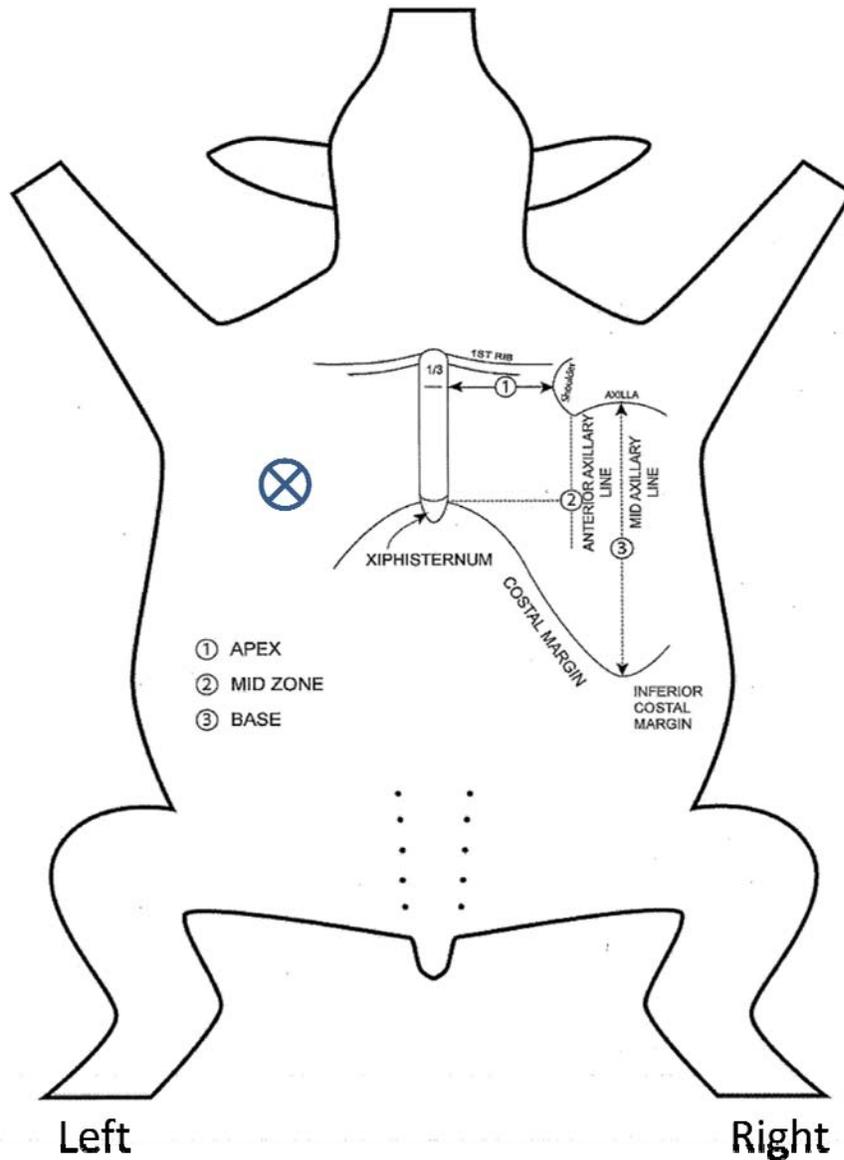
Figure 1. Lung recording positions on the left chest wall.

In experiment 2, the animals ($N = 7$) were evaluated during a progressive pneumothorax in the left hemi-thorax, with recordings taken after each successive increase in the size of the pneumothorax. The left pleural space was accessed by making a 5-cm incision between the ribs on the mid-axillary line and midway between the inferior costochondral margin and the sternal notch (incision site is marked with the letter "x"). An 18 Fr Foley catheter, covered with Vaseline gauze, secured with tape, and checked for air leaks, was placed into the pleural space as a chest tube. An initial pneumothorax was created by adding 150 ml of air to the left hemi-thorax. The volume of the pneumothorax was then increased to 300 ml and again to 500 ml. Throughout the experiment, the animal was monitored for changes in cardiovascular status (e.g., attenuated arterial pressure and cardiac output).

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For experiment 3, the animals were alternately divided into groups A ($n = 4$) and B ($n = 3$). Animals in group A received an additional 700 ml (total 1200 ml) of air into their left hemi-thorax to simulate a tension pneumothorax. Animals in group B received 250 ml of warm saline solution followed by an additional 250 ml of air injected into the pleural space to represent an increasing hemo-pneumothorax. The same cardiac and lung recordings were conducted following each intervention.

Evaluation of stethoscope returns

Cardiac function and ventilation were assessed by a investigator/flight surgeon using the acoustic and Doppler modes in turn. All acoustic returns were recorded for 30 seconds in digital format using Microsoft Waveform Audio File Format software and saved onto a Dell Latitude-300X laptop computer; subjective assessments were recorded on an Excel$^{©}$ spreadsheet by a medical technician familiar with both the recording equipment and the test device.

Acoustic breath sounds were assessed for:

a. Loudness rated as nil (0), quiet (1), moderate (2), or loud (3); assessments were subjective and relative to the previous experience of the investigator/flight surgeon.

b. Loudness relative to the contralateral thorax; right (R) or left (L) indicated which side was louder; equal (=) indicated that there was no discernable difference.

c. Additional qualities (e.g., gastric gurgle, tinkling, hollow, wheeze, squeak).

Doppler breath sounds were assessed for:

a. The presence and loudness of a previously noted distinctive low pitch rumble; assessments were again subjective relative to previous operator experience; quantitative measures used were nil (0), quiet (1), moderate (2), and loud (3).

b. The timing of the rumble, if notable (inspiratory, expiratory, or both).

c. Any interference from a vascular/cardiac source.

Results

The auscultation findings for each animal are reported in appendices A and B. Quantitative analyses for all animals are at appendices C and D.

The acoustic mode performed much like any other mechanical or electro-mechanical stethoscope although direct comparison with other models was not part of this experiment. The noise exclusion attributes were also not tested although it was notable that the sounds of an operating room (e.g., alarms, monitors, background noise) were still detectable through the NIS.

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The Doppler mode was always affected by significant "white noise" interference whether monitoring cardiac or ventilatory function. The interference appeared to have no consistent correlation with individual animals, site, position relative to the chest wall, movement of the device, gel quantity, or operator handling. The interference was hard to overcome and difficult to work with overall. However, a characteristic sound was heard in just over 30% of the Doppler test points. This sound occurred with the ventilatory cycle and was described as quiet, low pitched, and *rumbling* in nature. The positioning of the stethoscope head greatly affected detection of this characteristic sound; a minor adjustment in either placement or alignment of the stethoscope meant the difference in hearing the characteristic sound or not. The low pitched rumbling was rarely heard in the apices but was more common in the mid and lower lung fields.

Experiment 1 – Esophageal vs. Tracheal intubation

Acoustic sensor

Baseline acoustic returns were unremarkable except that returns were louder in the right apex and quieter in both bases. The misplacement of the ET tube in the esophagus caused loud and pathognomonic sounds such as a harsh gurgling or a metallic tinkling, easily audible using the acoustic mode of the NIS. *There was never any doubt that the ET tube was incorrectly situated.* In earlier animal models, the level of sedation was light, inducing some animals to start breathing spontaneously once they became sufficiently hypoxic. The additional sounds associated with these ventilatory efforts are noted in the results tables in appendix A.

Doppler sensor

The average scores for the Doppler rumble were marginally greater in the lung bases and with esophageal intubation (figure 2). However, the Doppler mode was never useful for diagnosing esophageal intubation in any of the animal models and it was always affected by significant white noise interference whether monitoring cardiac or ventilatory function.
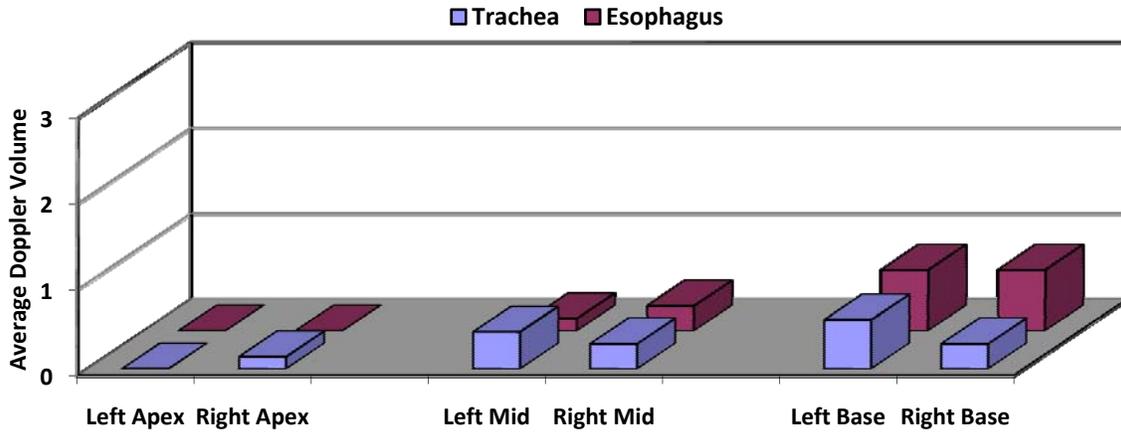
Figure 2. Doppler rumble volume (average scores, $N = 7$) with tracheal intubation
and esophageal intubation.

Experiment 2 – Detection of pneumothorax

Acoustic sensor

As the left-sided pneumothorax increased, there was a progressive reduction in audible
breath sounds in the left base, and, in one animal, a change to the pathognomonic breath sound
(hollow sounding). The findings are illustrated in figure 3.
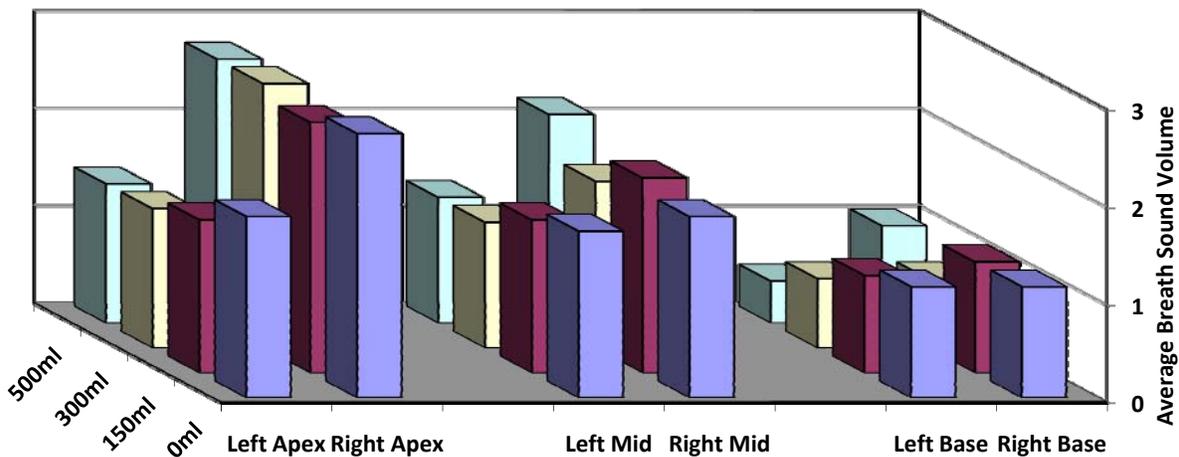
Figure 3. Breath sound volume (average scores, $N = 7$) compared with site and size
of a left-sided pneumothorax.

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Doppler returns were again assessed for the incidence and volume of the characteristic rumble. Acoustic returns were consistently below average across the entire lung fields, and especially more so in the apices which were very infrequent. *The loudest returns were heard in the right mid zone and more so with increasing pathology on the adjacent side*. Whilst a rumble was heard in the basal zones there was neither a difference between sides nor a relationship with the size of the pneumothorax. These findings are illustrated in figure 4.
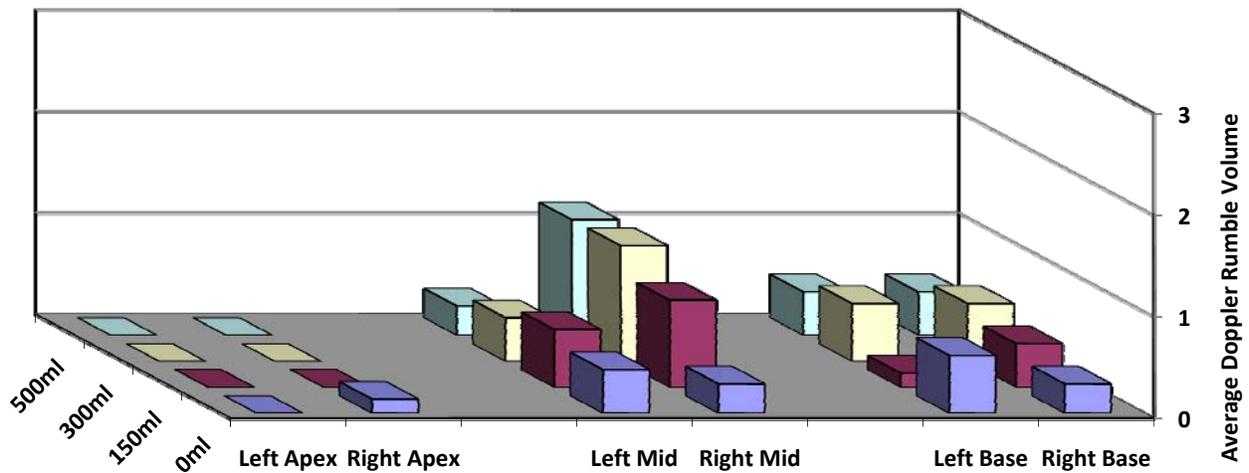
Figure 4. Doppler rumble volume (average scores, $N = 7$) compared with auscultation site and size of a left-sided pneumothorax.

If the volume of the characteristic Doppler return is disregarded with only the frequency of the characteristic rumble assessed (i.e., is it there or not?), the relationship is less pronounced in the mid zone but otherwise unchanged (figure 5).
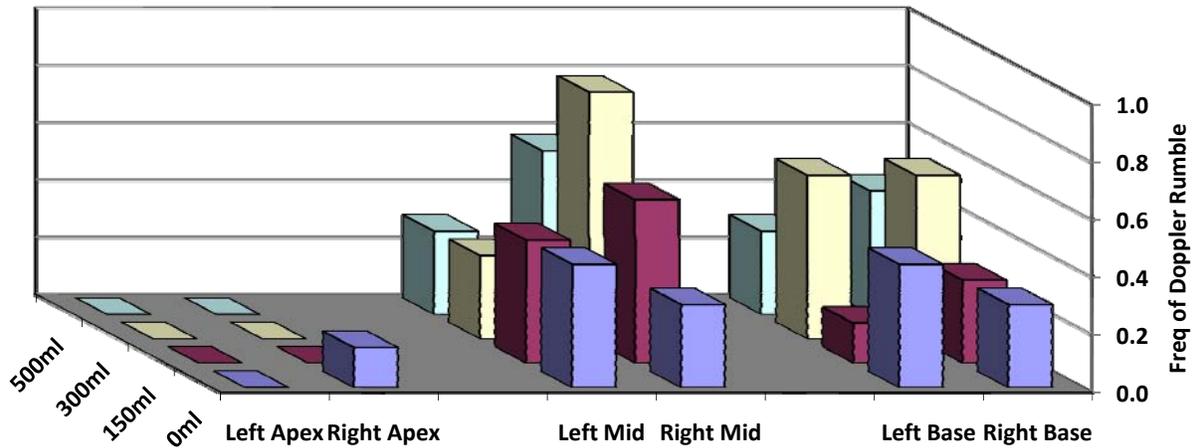
Figure 5. Frequency of Doppler rumble ($N = 7$) compared with auscultation site and size of a left-sided pneumothorax.

Experiment 3 – Detection of tension pneumothorax

Acoustic sensor

Four animals were progressed to a much larger pneumothorax (1200 ml). Acoustic findings (figure 6) developed no further than those previously established in experiment 2. The disparities in breath sound characteristics between each side were also no greater at 1200 ml than those findings at lesser volumes.
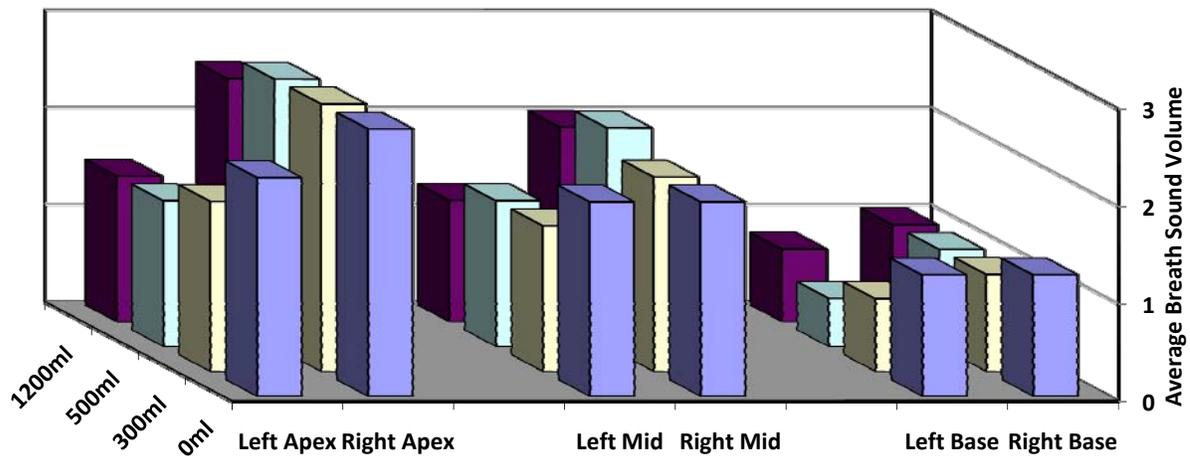
Figure 6. Breath sound volume (average scores, $n = 4$) compared with site and size of a left-sided tension pneumothorax.

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<u>Doppler sensor</u>

   The mid zone, with a 1200 ml pneumothorax, shows a greater disparity between opposing sides of the chest than that seen in experiment 2 (figure 7).
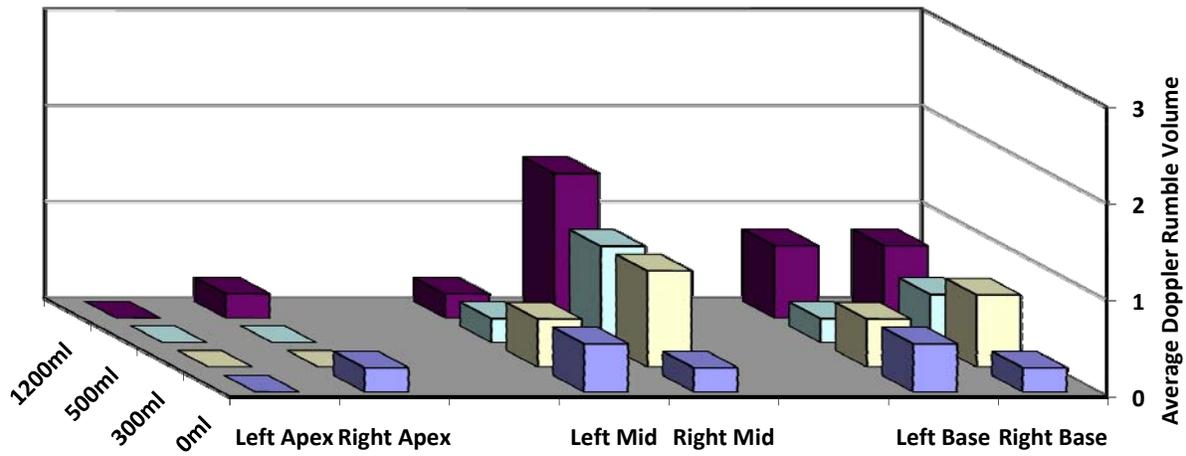
Figure 7. Doppler rumble volume (average scores, $n = 4$) compared with
         auscultation site and size of a left sided tension pneumothorax.

If the loudness of the characteristic rumble is discounted, with only frequency assessed, the trend is unchanged (figure 8).
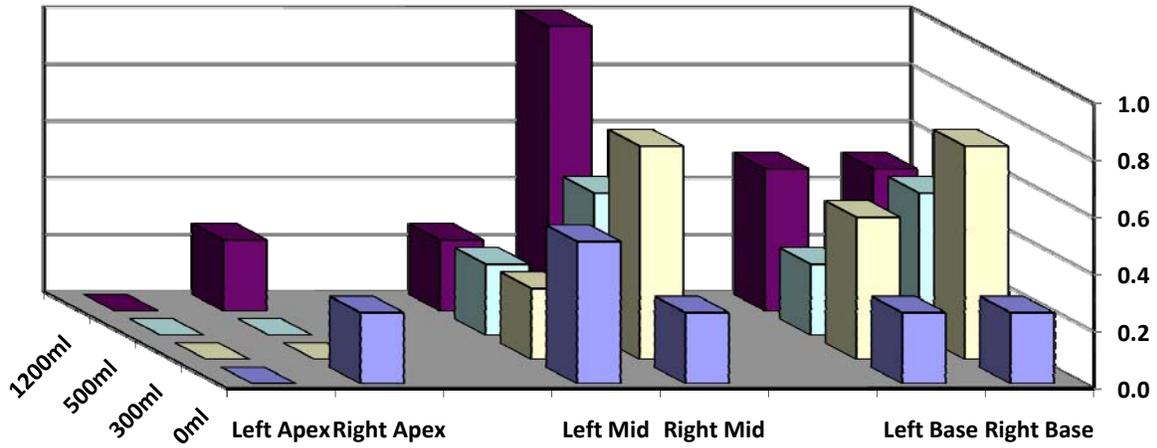
Figure 8. Doppler rumble frequency ($n = 4$) compared with auscultation site and size of a left sided tension pneumothorax.

Experiment 4 – Detection of hemo-pneumothorax

Acoustic sensor

Three animals were progressed to a hemo-pneumothorax using 250 ml of normal saline solution followed by another 250 ml of air. With such a low number of subjects, only gross differences were likely to have been evident as is shown in figure 9. There is limited evidence of any trend other than reducing acoustic returns in the basal zones as fluid is added to the pleural space. This is a bilateral finding and not in accordance with clinical expectation.
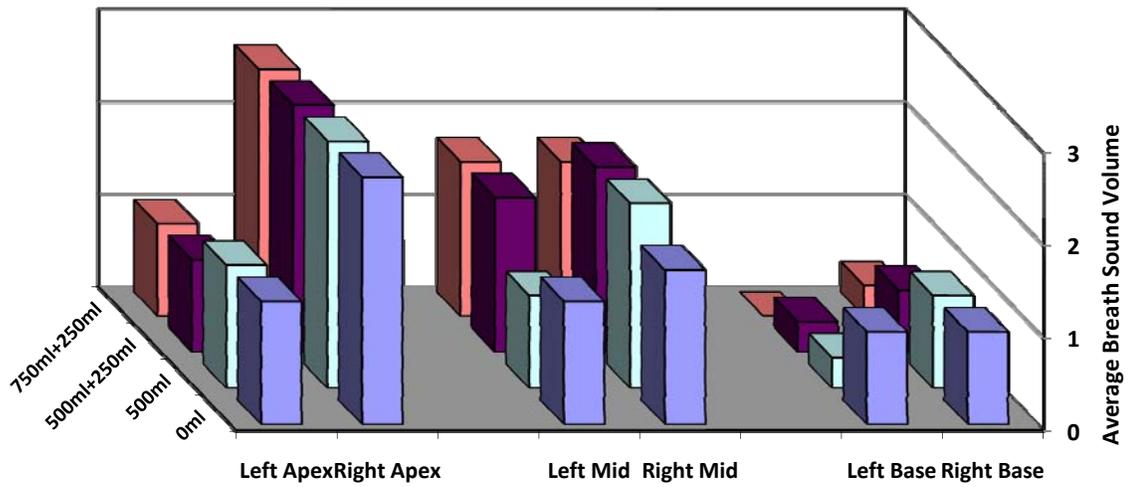
Figure 9. Breath sound volume (average scores, $n = 3$) compared with site and size of a left sided hemo-pneumothorax.

Doppler sensor

Whilst Doppler returns continued to be observed in the mid and basal zones, there was no overt pattern associated with the development of the hemo-pneumothorax when either the loudness or frequency of the sound was assessed.
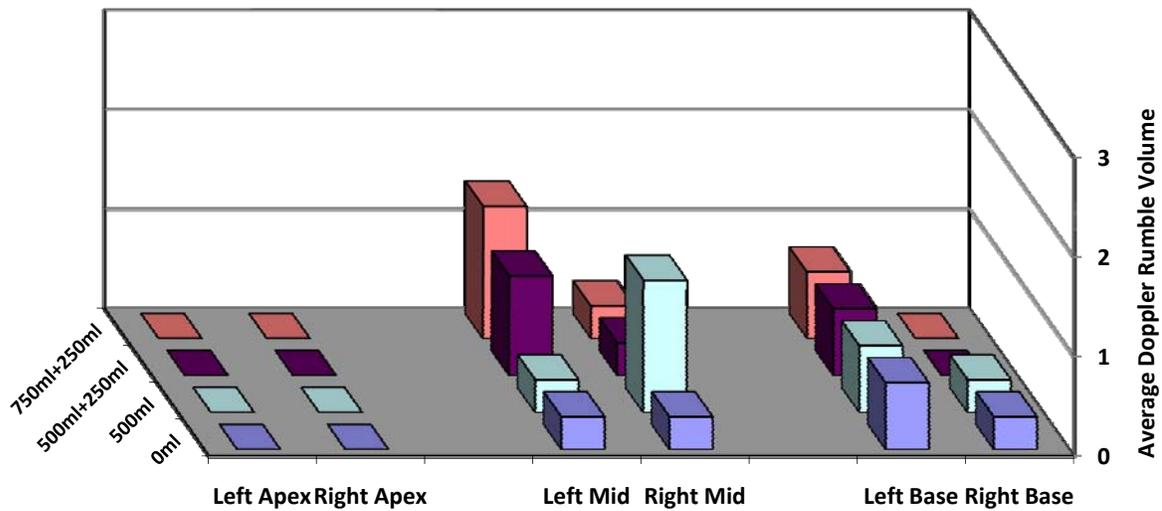
Figure 10. Doppler rumble volume (average scores, $n = 3$) compared with auscultation site and size of a left sided hemo-pneumothorax.

Discussion

The NIS was developed to enhance patient monitoring in high noise environments, such as a busy emergency room or within an evacuation vehicle. The acoustic mode has met with some acclaim when used by clinicians during military operations[2]. Insight into the Doppler mode, however, is sparse as there have been no formal trials examining its potential in patients with suspected chest pathology and most users have found the Doppler mode difficult to use and its returns difficult to interpret.[3] The noise immune qualities are, however, well-established following a trial in a noise simulator at the USAARL (Houtsma, Curry, Sewell, & Bernhard, 2006) and in-flight demonstrations (Houtsma & Curry, 2007), which proved the concept in healthy volunteers. For clinical acceptance, however, the NIS must be able to accurately detect vital signs; it must be able to distinguish disease with high sensitivity and specificity; it must be user friendly; it must be reasonably intuitive without extensive training; and learned skills should be sufficiently durable to avoid constant refresher training.

Acoustic mode

In this trial using swine models with three modes of induced pathology, the acoustic mode fared well. Findings (volume and qualitative assessments) were consistent with traditional auscultation when the ET tube was placed within the esophagus and when a pneumothorax was induced. Findings were less clear with the modeled hemo-pneumothorax, but this in part may be due to the low animal numbers in experiment 3, use of a swine model with a thick chest wall,

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[2] Personal communication Lt Col Alaistair Bushby / Dr. Bill Bernard, November 2008
[3] Personal communication: Lt Col Alaistair Bushby / Dr. Bill Bernhard / Capt Shaun Westphal / Dr. Shawn Kane, November 2008.

and/or the inability to readily monitor the posterior chest wall in the anaesthetized animal in a supine position (the infused saline is likely to have tracked down to the dependent parts of the chest cavity, i.e., posteriorly in the supine animal).

Nevertheless, it is likely that the elicited signs would be sufficient to reach accurate diagnosis particularly if more auscultation sites were used and findings were compared with the contralateral side. There was no direct comparison with alternative devices and thus no conclusion on relative merit is possible. It is, however, worth mentioning that significant ambient noise is still detectable through the NIS via noise assimilation directly into the animal chest cavity and this is always likely to be a limitation of this mode within high noise environments.

Two other acoustic findings are worthy of deliberation. Throughout the experiment, the right apex gave consistently louder acoustic returns whilst the bases gave relatively quiet returns. No explanation is offered for this finding other than the anatomy of the thoracic space within the swine model. Secondly, development of a much larger (1200 ml) pneumothorax did not develop the clinical findings significantly beyond those noted at 500 ml. It is possible that there was some leakage of gas around the catheter insertion site although the purse string suture should have prevented this. At the end of each series and prior to euthanasia, gas was drawn out of the pleural space and measured – this usually came to within 75% of the injected amount suggesting some leakage or dispersion within the chest cavity.

Doppler mode

Definitive Doppler findings were difficult to elicit using the stethoscope provided for this study, and appeared neither sensitive to, nor specific of the induced pathology. The only sound associated with the ventilatory cycle was the low pitched rumble, but this was difficult to elicit from one ventilatory cycle to another and found in less than one third of all measurement series. It was most commonly heard in the mid zones (47% of recordings) and lung bases (43% of recordings); it was rarely heard in the apices (2% of recordings). The rumble was always much quieter than the comparable acoustic returns. It is probable that the predictability of the ventilatory cycle in the anaesthetized animal made it easier to detect than in a subject breathing spontaneously and with less regularity.

Successful detection was extremely susceptible to small movements of the Doppler head implying the reflective surface was rather small. It is also necessary for the reflective surface to move to develop a positive Doppler return and it is unclear what fluids or structures might fulfill this within lung tissue other than large blood vessels. Whilst these are prominent in the hilar regions of the lung, they are unlikely to experience gross dynamic changes with an esophageal ET tube or the development of pneumothorax. The pleural surfaces are sufficient to reflect an ultrasound beam but move little relative to the Doppler head even when displaced by a pneumothorax or hemothorax and thus would not be expected to generate a significant Doppler return either.

The characteristic Doppler rumble was heard in all of the animals – normal, esophageal ET tube, pneumothorax and haemo-pneumothorax. Whilst the rumble may vary in volume, it never

changes its qualitative nature. In other words, the returns are relatively binary (i.e., they are there or not) which reduces their predictive power as a diagnostic tool. By contrast, returns from the acoustic mode vary in character, and may even be pathognomonic, thus aiding diagnosis.

However the Doppler studies were not without some positive findings. Firstly the low pitch rumble was noted more frequently in the basal zones with esophageal intubation. There is no clear explanation for this but it may relate to the timing of the Doppler assessments; as Doppler examination of the basal zones was always the final part of the examination sequence this finding may be related to the increased ventilatory effort or cardiac output of some animals as they became progressively more hypoxic. Secondly, development of a pneumothorax caused the low pitched rumble to occur more frequently and in greater volume on the *contralateral* side in the mid zone. This finding was particularly notable with development of the simulated tension pneumothorax (1200 ml). This may represent a shift in the mediastinum enabling increased reflectance of the ultrasound beam or a change in the haemodynamics within the chest cavity.

Test instrument considerations

Interference was a major limitation with Doppler function in this NIS model. Whether in the acoustic mode or Doppler mode, both settings were similarly afflicted. The authors were unable to identify any provocative or protective factors and are thus unable to determine causation. Involvement of technicians and clinicians experienced with ultrasound and Doppler may be able to identify the predominant sources of the interference and suggest means of control. For instance, it may be possible to selectively filter out the majority of the interference, it being of much higher frequency. Secondly, the lack of an on-off switch is a mild inconvenience – the timer occasionally cut off an examination in mid flow; conversely when the operator needed to communicate with others, then the NIS had to be disconnected which is rather less convenient than a simple on-off switch. Finally alternating between acoustic and Doppler modes requires gel to be either added or removed which, although necessary, is irksome. More elegant solutions would make the device rather more user friendly particularly if used in austere circumstances.

Study design limitations

The following are suggested as study limitations:

a. The swine chest wall is thick, muscular, and has closely aligned ribs. The transference of ultrasound waves is heavily influenced by dense tissue; it is probable that Doppler returns were degraded and interference made worse as a result. Future studies should involve humans or utilize animals with a slighter chest wall if considering interventional studies.

b. The pig chest wall also features a very angular sternal ridge and parasternal areas which made stable placement of the large NIS head difficult whilst monitoring cardiac returns. The monitoring site had to be adjusted from one animal to another to attain satisfactory cardiac returns. It was also notable that the cardiac Doppler returns became progressively harder to elucidate toward the end of each animal series. Explanations for this are unclear unless it is related to the prolonged immobility of the anesthetized animal in a supine position. However the

cardiac returns were not a vital part of the study and this finding did not significantly detract from the study objectives.

c. When saline solution is injected into the pleural space it will track toward the most dependent area; in this case, the posterior pleural space. Unfortunately, this was not accounted for when monitoring sites were established, in part because the animal is securely tied to the operating table and thus, relatively immobile. Consequently the development of a hemo-pneumothorax was likely to have had little influence on the findings due to predetermined monitoring sites. Future studies investigating similar interventions should account for this.

d. Previous observations using the NIS on human patients with established chest trauma have included the discovery of novel sounds, e.g., the "whip-whistle" noted in patients with a traumatic pneumothorax.[4] Traumatic chest injuries frequently induce damage to parietal and visceral pleura, and underlying lung tissue. In this study only the parietal pleura was breached with no damage to the underlying visceral pleura or lung tissue. Consequently there was no air leakage from lung tissue which may be responsible for this previously noted novel sound. It is hard to induce such pathology in a controlled and measurable manner in an interventional study such as this. However, further human studies on trauma patients may help to evaluate this aspect further.

Conclusions

The acoustic mode performed well in this study although it is evident that it will always be affected by ambient noise, as the instrument and the chest cannot be totally isolated from unwanted noise. The Doppler mode is an interesting development but is limited in a diagnostic sense in its current format. It is blighted by interference, relatively difficult to detect and the characteristic rumble is neither sensitive to nor specific of individual disease states. However, Doppler did produce positive findings in relation to the development of a pneumothorax albeit on the contralateral side.

Recommendations

a. Further development of the NIS requires a clear understanding of Doppler compatible reflective surfaces within the chest cavity and the origins of the intrusive interference. Involvement of cardiologists and technicians familiar with echocardiography may be supportive.

b. Formal studies in human trauma and diseases cases are required to determine the diagnostic value of the Doppler and acoustic modes. These studies are currently underway.

c. If the NIS becomes established as a useful diagnostic tool, then incorporation of an on-off switch and alternatives to ultrasound gel (e.g., semi solid gels as used in a stand-off) may enhance utility in challenging environments.

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[4] Personal communication Lt Col Alaistair Bushby / Dr. Bill Bernard, November 2008.

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Acoustics results.

709	Left apex	Right apex	Left Mid	Right Mid	Left Base	Right Base	Cardiac
Baseline Acoustic	MODERATE	MODERATE	MODERATE	MODERATE	MODERATE	MODERATE	
Esophageal Acoustic	MODERATE GASTRIC GURGLE	MODERATE GASTRIC GURGLE	MODERATE GASTRIC GURGLE	MODERATE GASTRIC GURGLE	QUIET GASTRIC GURGLE	QUIET GASTRIC GURGLE	N/A
Pneumo 150 Acoustic	MODERATE	MODERATE	MODERATE	MODERATE	QUIET	MODERATE	
Pneumo 300 Acoustic	MODERATE	MODERATE	QUIET	MODERATE	NIL	QUIET	
Pneumo 500 Acoustic	QUIET	MODERATE	QUIET	MODERATE	NIL	QUIET	
Pneumo 1200 Acoustic	MODERATE, HOLLOW, LEAK	MODERATE	QUIET	MODERATE	NIL	QUIET	

126	Left apex	Right apex	Left Mid	Right Mid	Left Base	Right Base	Cardiac
Baseline Acoustic	QUIET	MODERATE	QUIET	QUIET	QUIET	QUIET	
Esophageal Acoustic-	LOUD GASTRIC GURGLE	LOUD GASTRIC GURGLE	LOUD GASTRIC GURGLE	LOUD GASTRIC GURGLE, BREATH SOUNDS	LOUD GASTRIC GURGLE	LOUD GASTRIC GURGLE	N/A
Pneumo 150 Acoustic	QUIET	MODERATE	QUIET	MODERATE	QUIET	QUIET	
Pneumo 300 Acoustic	QUIET	MODERATE	QUIET	QUIET	QUIET	NIL	
Pneumo 500 Acoustic	QUIET	MODERATE	QUIET	MODERATE	NIL	QUIET	
Hemo 250 Acoustic	QUIET	MODERATE	QUIET	MODERATE	NIL	QUIET	
Hemo 250/ Pneumo 250 Acoustic	QUIET, SQUEAK	MODERATE	MODERATE	QUIET	NIL	NIL	

137	Left apex	Right apex	Left Mid	Right Mid	Left Base	Right Base	Cardiac
Baseline Acoustic							
Baseline Doppler	NIL	NIL	NIL	NIL	NIL	RUMBLE	
CO2 DECREASED FROM 38 TO 21 AT 1005. CARDIOVASCULAR COLLAPSE AT 1006.							

135	Left apex	Right apex	Left Mid	Right Mid	Left Base	Right Base	Cardiac
Baseline Acoustic	MODERATE	LOUD R	MODERATE	MODERATE R	QUIET	QUIET =	MURMUR
Esophageal Acoustic	QUIET GASTRIC GURGLE	LOUD GASTRIC GURGLE	LOUD GASTRIC GURGLE, BREATH SOUNDS	LOUD GASTRIC GURGLE	GASTRIC GURGLE, BREATH SOUNDS	LOUD GASTRIC GURGLE, BREATH SOUNDS	N/A
Pneumo 150 Acoustic	MODERATE	LOUD R	MODERATE	LOUD R	QUIET	QUIET R	
Pneumo 300 Acoustic	MODERATE	LOUD R	MODERATE	MODERATE R	QUIET	QUIET =	
Pneumo 500 Acoustic	MODERATE	LOUD R	MODERATE	LOUD R	QUIET	QUIET R	
Pneumo 1200 Acoustic	QUIET	LOUD R	QUIET	MODERATE R	QUIET, HOLLOW	QUIET R	

128	Left apex	Right apex	Left Mid	Right Mid	Left Base	Right Base	Cardiac
Baseline Acoustic	QUIET	LOUD	MODERATE	MODERATE =	QUIET	QUIET =	
Esophageal Acoustic	LOUD GASTRIC GURGLE	LOUD GASTRIC GURGLE	MODERATE GASTRIC GURGLE	GASTRIC TINKLING, BREATH SOUNDS	GASTRIC TINKLING, BREATH SOUNDS	GASTRIC TINKLING, BREATH SOUNDS	N/A
Pneumo 150 Acoustic	QUIET	LOUD R	QUIET	MODERATE R	QUIET	QUIET =	CLICKS
Pneumo 300 Acoustic	QUIET, FAINT SQUEAK	LOUD R	QUIET, INSPIRATORY SQUEAK	QUIET R, INSPIRATORY SQUEAK	QUIET	QUIET R	
Pneumo 500 Acoustic	QUIET	LOUD R	QUIET	MODERATE R	QUIET	QUIET =	
Hemo 250 Acoustic	QUIET	LOUD R	MODERATE	MODERATE =	QUIET	QUIET R	INTERMITTENT CLICK
Hemo/Pneumo Acoustic	QUIET	LOUD R	QUIET	MODERATE R	NIL	NIL	

127	Left apex	Right apex	Left Mid	Right Mid	Left Base	Right Base	Cardiac
Baseline Acoustic	LOUD	LOUD R	MODERATE	MODERATE =	QUIET	QUIET =	
Esophageal Acoustic	QUIET GASTRIC GURGLE	MODERATE GASTRIC GURGLE, BREATH SOUNDS	QUIET GASTRIC GURGLE, BREATH SOUNDS	MODERATE GASTRIC GURGLE, BREATH SOUNDS	MODERATE GASTRIC GURGLE, BREATH SOUNDS	MODERATE GASTRIC GURGLE, BREATH SOUNDS	N/A
Pneumo 150 Acoustic	MODERATE	MODERATE R	QUIET	QUIET=	QUIET	QUIET =	RESPIRATORY SQUEAK
Pneumo 300 Acoustic	QUIET	LOUD R	QUIET	MODERATE R	QUIET	QUIET R	
Pneumo 500 Acoustic	QUIET	LOUD R	QUIET	MODERATE R	NIL	QUIET R	
Pneumo 1200 Acoustic	QUIET	LOUD R	QUIET	MODERATE R	QUIET	QUIET R	

131	Left apex	Right apex	Left Mid	Right Mid	Left Base	Right Base	Cardiac
Baseline Acoustic	MODERATE	LOUD R	QUIET	MODERATE R	QUIET	QUIET L	CLICK
Esophageal Acoustic	LOUD GASTRIC GURGLE	VERY LOUD GASTRIC GURGLE	LOUD GASTRIC GURGLE & TINKLING	MODERATE GASTRIC GURGLE & TINKLING	LOUD GASTRIC GURGLE & TINKLING	LOUD GASTRIC GURGLE & TINKLING	
Pneumo 150 Acoustic	QUIET	LOUD R	MODERATE	MODERATE R	QUIET	QUIET =	
Pneumo 300 Acoustic	QUIET	LOUD R	QUIET	MODERATE R	NIL	NIL	
Pneumo 500 Acoustic	MODERATE	LOUD R	QUIET, INSPIRATORY WHEEZE	MODERATE R	NIL	QUIET R	INSP BURBLE
Hemo 250 Acoustic	QUIET	LOUD R	MODERATE	MODERATE R	NIL	NIL	
Hemo 250/ Pneumo 250 Acoustic	QUIET, RESPIRATORY FIZZ	LOUD R	MODERATE	MODERATE =	NIL	QUIET R	

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132	Left apex	Right apex	Left Mid	Right Mid	Left Base	Right Base	Cardiac
Baseline Acoustic	MODERATE	LOUD R	MODERATE	MODERATE =	QUIET	QUIET L	
Esophageal Acoustic	SHORT GASTRIC GURGLE	SHORT GASTRIC GURGLE, BREATH SOUNDS	SHORT GASTRIC GURGLE, SNORING BREATH SOUNDS	SHORT GASTRIC GURGLE, SNORING BREATH SOUNDS	SHORT GASTRIC GURGLE, SNORING BREATH SOUNDS	SHORT GASTRIC GURGLE, SNORING BREATH SOUNDS	N/A
Pneumo 150 Acoustic	MODERATE, INSPIRATORY SQUEAK	LOUD R, INSPIRATORY SNORT, VENTILATOR SOUNDS	MODERATE, INSPIRATORY SQUEAK, VENTILATOR SOUNDS	MODERATE =, INSPIRATORY SQUEAK, VENTILATOR SOUNDS	QUIET, INSPIRATORY SQUEAK, VENTILATOR SOUNDS	QUIET L, VENTILATOR SOUNDS	
Pneumo 300 Acoustic	MODERATE, EXPIRATORY BLOW	LOUD R	MODERATE	MODERATE =	QUIET	QUIET L	
Pneumo 500 Acoustic	MODERATE, EXPIRATORY SNORT	LOUD R, INSPIRATORY SNORT	MODERATE, INSPIRATORY SNORT	MODERATE =, INSPIRATORY SNORT	QUIET, INSPIRATORY SNORT	QUIET =	
Pneumo 1200 Acoustic	MODERATE, INSPIRATORY SNORT	LOUD R, INSPIRATORY SNORT	MODERATE, INSPIRATORY SNORT	MODERATE =, INSPIRATORY SNORT	QUIET, INSPIRATORY SNORT	QUIET L, FAINT INSPIRATORY SNORT	

Appendix B.

Doppler results.

709	Left apex	Right apex	Left Mid	Right Mid	Left Base	Right Base	Cardiac
Baseline Doppler	CARDIAC SOUNDS	NIL	QUIET RUMBLE	NIL	MODERATE RUMBLE	QUIET RUMBLE	
Esophageal Doppler	CARDIAC SOUNDS	CARDIAC SOUNDS	NIL	NIL	QUIET RUMBLE	QUIET RUMBLE	N/A
Pneumo 150 Doppler	NIL	NIL	QUIET RUMBLE	QUIET RUMBLE	QUIET RUMBLE	MODERATE RUMBLE	
Pneumo 300 Doppler	NIL	NIL	INTERMITTENT CARDIAC SOUNDS	QUIET RUMBLE	QUIET RUMBLE, INSPIRATORY	QUIET RUMBLE	
Pneumo 500 Doppler	NIL	NIL	NIL	NIL	NIL	QUIET RUMBLE	
Pneumo 1200 Doppler	NIL	QUIET RUMBLE	NIL	QUIET RUMBLE	MODERATE RUMBLE	NIL	QUIET

126	Left apex	Right apex	Left Mid	Right Mid	Left Base	Right Base	Cardiac
Baseline Doppler	NIL	NIL	NIL	NIL	QUIET RUMBLE, EXPIRATORY	NIL	
Esophageal Doppler (Off ventilator breathing)	NIL	NIL	NIL	NIL	MODERATE RUMBLE	MODERATE RUMBLE	N/A
Pneumo 150 Doppler	NIL	NIL	NIL	NIL	NIL	NIL	
Pneumo 300 Doppler	NIL	NIL	NIL	MODERATE RUMBLE, INSPIRATORY AND EXPIRATORY	NIL	NIL	
Pneumo 500 Doppler	NIL	NIL	NIL	NIL	NIL	NIL	
Hemo 250 Doppler	NIL	NIL	QUIET RUMBLE	NIL	QUIET RUMBLE	NIL	
Hemo 250 /Pneumo 250 Doppler	NIL	NIL	MODERATE RUMBLE	NIL	MODERATE RUMBLE	NIL	

135	Left apex	Right apex	Left Mid	Right Mid	Left Base	Right Base	Cardiac
Baseline Doppler	NIL	QUIET RUMBLE	NIL	NIL	NIL	NIL	
Esophageal Doppler	NIL	NIL	NIL	NIL	NIL	NIL	N/A
Pneumo 150 Doppler	NIL	NIL	NIL	NIL	NIL	QUIET RUMBLE	
Pneumo 300 Doppler	NIL	NIL	NIL	NIL	NIL	QUIET RUMBLE	
Pneumo 500 Doppler	NIL	NIL	NIL	NIL	NIL	NIL	
Pneumo 1200 Doppler	NIL	NIL	NIL	QUIET RUMBLE	NIL	QUIET RUMBLE	

128	Left apex	Right apex	Left Mid	Right Mid	Left Base	Right Base	Cardiac
Baseline Doppler	NIL	NIL	NIL	NIL	NIL	QUIET RUMBLE	
Esophageal Doppler	NIL	NIL	NIL	NIL	NIL	NIL	N/A
Pneumo 150 Doppler	NIL	NIL	NIL	QUIET RUMBLE	NIL	ABNORMAL SOUNDS	
Pneumo 300 Doppler	NIL	NIL	QUIET RUMBLE, EXPIRATORY	QUIET RUMBLE, EXPIRATORY	QUIET RUMBLE, INSPIRATORY & EXPIRATORY	QUIET RUMBLE, EXPIRATORY	
Pneumo 500 Doppler	NIL	NIL	NIL	MODERATE RUMBLE, INSPIRATORY & EXPIRATORY	NIL	QUIET RUMBLE, EXPIRATORY	
Hemo 250 Doppler	NIL	NIL	NIL, CARDIAC SOUNDS IN RESPIRATORY CYCLE	QUIET RUMBLE, EXPIRATORY	NIL	NIL	
Hemo/Pneumo Doppler	NIL	NIL	QUIET RUMBLE, EXPIRATORY	QUIET RUMBLE, EXPIRATORY	NIL	NIL	

127	Left apex	Right apex	Left Mid	Right Mid	Left Base	Right Base	Cardiac
Baseline Doppler	NIL	NIL	NIL	NIL	NIL	NIL	
Esophageal Doppler		NIL	NIL	NIL	QUIET RUMBLE, CHEST WALL EFFORT NOT RELATED TO VENTILATOR	QUIET RUMBLE, CHEST WALL EFFORT NOT RELATED TO VENTILATOR	N/A
Pneumo 150 Doppler	NIL	NIL	NIL	QUIET RUMBLE	NIL	NIL	
Pneumo 300 Doppler	NIL	NIL	CYCLICAL CARDIAC SOUNDS	QUIET RUMBLE	QUIET RUMBLE	QUIET RUMBLE	
Pneumo 500 Doppler	NIL	NIL	NIL	MODERATE RUMBLE	QUIET RUMBLE	QUIET RUMBLE	
Pneumo 1200 Doppler	NIL	NIL	NIL	MODERATE RUMBLE	QUIET RUMBLE	MODERATE RUMBLE	

131	Left apex	Right apex	Left Mid	Right Mid	Left Base	Right Base	Cardiac
Baseline Doppler	NIL	NIL	QUIET RUMBLE	QUIET RUMBLE	QUIET RUMBLE	NIL	
Esophageal Doppler	NIL	NIL	NIL	QUIET RUMBLE	QUIET RUMBLE*	QUIET RUMBLE*	
Pneumo 150 Doppler	NIL	NIL	QUIET RUMBLE	NIL	NIL	NIL	
Pneumo 300 Doppler	NIL	NIL	NIL	QUIET RUMBLE	QUIET RUMBLE, ABNORMAL	NIL	
Pneumo 500 Doppler	NIL	NIL	QUIET RUMBLE	MODERATE RUMBLE	MODERATE RUMBLE, ABNORMAL	NIL	
Hemo 250 Doppler	NIL	NIL	MODERATE RUMBLE	NIL	QUIET RUMBLE	NIL	
Hemo 250 /Pneumo 250 Doppler	NIL	RESPIRATORY VARIATION TO CARDIAC SOUND	QUIET RUMBLE, INTERMITTENT	NIL	NIL	NIL	

*MODERATE RUMBLE ASSOCIATED WITH CHEST WALL EFFORT NOT RELATED TO VENTILATOR

132	Left apex	Right apex	Left Mid	Right Mid	Left Base	Right Base	Cardiac
Baseline Doppler	NIL	NIL	QUIET RUMBLE	QUIET RUMBLE	NIL	NIL	
Esophageal Doppler	NIL	NIL	RUMBLE WITH CHEST WALL MOVEMENT	RUMBLE WITH CHEST WALL MOVEMENT	RUMBLE WITH CHEST WALL MOVEMENT	RUMBLE WITH CHEST WALL MOVEMENT	N/A
Pneumo 150 Doppler	NIL	NIL	MODERATE RUMBLE	LOUD RUMBLE	NIL	NIL	
Pneumo 300 Doppler	NIL, PROMINENT CARDIAC SOUNDS	NIL	MODERATE RUMBLE	MODERATE RUMBLE	NIL	NIL	
Pneumo 500 Doppler	NIL	NIL	QUIET RUMBLE	MODERATE RUMBLE	NIL	NIL	
Pneumo 1200 Doppler	NIL	NIL	QUIET RUMBLE	MODERATE RUMBLE	NIL	NIL	

Appendix C.

Acoustics scores.

Figures shaded in gray indicate acoustic returns were subjectively louder than contralateral side.

709	Left apex	Right apex	Left Mid	Right Mid	Left Base	Right Base
Baseline Acoustic	2	2	2	2	2	2
Esophageal Acoustic						
Pneumo 150 Acoustic	2	2	2	2	1	2
Pneumo 300 Acoustic	2	2	1	2	0	1
Pneumo 500 Acoustic	1	2	1	2	0	1
Pneumo 1200 Acoustic	2	2	1	2	0	1
Hemo 250 Acoustic						
Hemo 250 / Pneumo 250 Acoustic						

126	Left apex	Right apex	Left Mid	Right Mid	Left Base	Right Base
Baseline Acoustic	1	2	1	1	1	1
Esophageal Acoustic						
Pneumo 150 Acoustic	1	2	1	2	1	1
Pneumo 300 Acoustic	1	2	1	1	1	0
Pneumo 500 Acoustic	1	2	1	2	0	1
Pneumo 1200 Acoustic						
Hemo 250 Acoustic	1	2	1	2	0	1
Hemo 250 / Pneumo 250 Acoustic	1	2	2	1	0	0

135	Left apex	Right apex	Left Mid	Right Mid	Left Base	Right Base
Baseline Acoustic	2	3	2	2	1	1
Esophageal Acoustic						
Pneumo 150 Acoustic	2	3	2	3	1	1
Pneumo 300 Acoustic	2	3	2	2	1	1
Pneumo 500 Acoustic	2	3	2	3	1	1
Pneumo 1200 Acoustic	1	2	1	2	1	1
Hemo 250 Acoustic						
Hemo 250 / Pneumo 250 Acoustic						

128	Left apex	Right apex	Left Mid	Right Mid	Left Base	Right Base
Baseline Acoustic	1	3	2	2	1	1
Esophageal Acoustic						
Pneumo 150 Acoustic	1	3	1	2	1	1
Pneumo 300 Acoustic	1	3	1	1	1	1
Pneumo 500 Acoustic	1	3	1	2	1	1
Pneumo 1200 Acoustic						
Hemo 250 Acoustic	1	3	2	2	1	1
Hemo 250 / Pneumo 250 Acoustic	1	3	1	2	0	0

127	Left apex	Right apex	Left Mid	Right Mid	Left Base	Right Base
Baseline Acoustic	3	3	2	2	1	1
Esophageal Acoustic						
Pneumo 150 Acoustic	2	2	1	1	1	1
Pneumo 300 Acoustic	1	3	1	2	1	1
Pneumo 500 Acoustic	1	3	1	2	0	1
Pneumo 1200 Acoustic	1	3	1	2	1	1
Hemo 250 Acoustic						
Hemo 250 / Pneumo 250 Acoustic						

131	Left apex	Right apex	Left Mid	Right Mid	Left Base	Right Base
Baseline Acoustic	2	3	1	2	1	1
Esophageal Acoustic						
Pneumo 150 Acoustic	1	3	2	2	1	1
Pneumo 300 Acoustic	1	3	1	2	0	0
Pneumo 500 Acoustic	2	3	1	2	0	1
Pneumo 1200 Acoustic						
Hemo 250 Acoustic	1	3	2	2	0	0
Hemo 250 / Pneumo 250 Acoustic	1	3	2	2	0	1

132	Left apex	Right apex	Left Mid	Right Mid	Left Base	Right Base
Baseline Acoustic	2	3	2	2	1	1
Esophageal Acoustic						
Pneumo 150 Acoustic	2	3	2	2	1	1
Pneumo 300 Acoustic	2	3	2	2	1	1
Pneumo 500 Acoustic	2	3	2	2	1	1
Pneumo 1200 Acoustic	2	3	2	2	1	1
Hemo 250 Acoustic						
Hemo 250 / Pneumo 250 Acoustic						

ALL	Left apex	Right apex	Left Mid	Right Mid	Left Base	Right Base
Baseline Acoustic	13	19	12	13	8	8
Pneumo 150 Acoustic	11	18	11	14	7	8
Pneumo 300 Acoustic	10	19	9	12	5	5
Pneumo 500 Acoustic	10	19	9	15	3	7
Pneumo 1200 Acoustic	6	10	5	8	3	4
Hemo 250 Acoustic	3	8	5	6	1	2
Hemo 250 / Pneumo 250 Acoustic	3	8	5	5	0	1

TENSION SERIES	Left apex	Right apex	Left Mid	Right Mid	Left Base	Right Base
Baseline Acoustic	9	11	8	8	5	5
Pneumo 150 Acoustic	8	10	7	8	4	5
Pneumo 300 Acoustic	7	11	6	8	3	4
Pneumo 500 Acoustic	6	11	6	9	2	4
Pneumo 1200 Acoustic	6	10	5	8	3	4

HEMO/PNEUMO SERIES	Left apex	Right apex	Left Mid	Right Mid	Left Base	Right Base
Baseline Acoustic	4	8	4	5	3	3
Pneumo 150 Acoustic	3	8	4	6	3	3
Pneumo 300 Acoustic	3	8	3	4	2	1
Pneumo 500 Acoustic	4	8	3	6	1	3
Hemo 250 Acoustic	3	8	5	6	1	2
Hemo 250 / Pneumo 250 Acoustic	3	8	5	5	0	1

Doppler scores.

709	Left apex	Right apex	Left Mid	Right Mid	Left Base	Right Base
Baseline Doppler			1		2	1
Esophageal Doppler					1	1
Pneumo 150 Doppler			1	1	1	2
Pneumo 300 Doppler				1	1	1
Pneumo 500 Doppler						1
Pneumo 1200 Doppler		1		1	2	

126	Left apex	Right apex	Left Mid	Right Mid	Left Base	Right Base
Baseline Doppler					1	
Esophageal Doppler					2	2
Pneumo 150 Doppler						
Pneumo 300 Doppler				2		
Pneumo 500 Doppler						
Hemo 250 Doppler			1		1	
Hemo 250/Pneumo 250 Doppler			2		2	

135	Left apex	Right apex	Left Mid	Right Mid	Left Base	Right Base
Baseline Doppler		1				
Esophageal Doppler						
Pneumo 150 Doppler						1
Pneumo 300 Doppler						1
Pneumo 500 Doppler						
Pneumo 1200 Doppler				1		1

128	Left apex	Right apex	Left Mid	Right Mid	Left Base	Right Base
Baseline Doppler						1
Esophageal Doppler						
Pneumo 150 Doppler				1		
Pneumo 300 Doppler			1	1	1	1
Pneumo 500 Doppler				2		1
Hemo 250 Doppler				1		
Hemo 250 /Pneumo 250 Doppler			1	1		

127	Left apex	Right apex	Left Mid	Right Mid	Left Base	Right Base
Baseline Doppler						
Esophageal Doppler					1	1
Pneumo 150 Doppler				1		
Pneumo 300 Doppler				1	1	1
Pneumo 500 Doppler				2	1	1
Pneumo 1200 Doppler				2	1	2

131	Left apex	Right apex	Left Mid	Right Mid	Left Base	Right Base
Baseline Doppler			1	1	1	
Esophageal Doppler				1	1	1
Pneumo 150 Doppler			1			
Pneumo 300 Doppler				1	1	
Pneumo 500 Doppler			1	2	2	
Hemo 250 Doppler			2		1	
Hemo 250/Pneumo 250 Doppler			1			

132	Left apex	Right apex	Left Mid	Right Mid	Left Base	Right Base
Baseline Doppler			1	1		
Esophageal Doppler			1	1	1	1
Pneumo 150 Doppler			2	3		
Pneumo 300 Doppler			2	2		
Pneumo 500 Doppler			1	2		
Pneumo 1200 Doppler			1	2		

ALL ANIMALS	Left apex	Right apex	Left Mid	Right Mid	Left Base	Right Base
Baseline Doppler	0	1	3	2	4	2
Pneumo 150 Doppler	0	0	4	6	1	3
Pneumo 300 Doppler	0	0	3	8	4	4
Pneumo 500 Doppler	0	0	2	8	3	3
Pneumo 1200 Doppler	0	1	1	6	3	3
Hemo 250 Doppler	0	0	3	1	2	0
Hemo 250/Pneumo 250 Doppler	0	0	4	1	2	0

TENSION SERIES ONLY	Left apex	Right apex	Left Mid	Right Mid	Left Base	Right Base
Baseline Doppler	0	1	2	1	2	1
Pneumo 150 Doppler	0	0	3	5	1	3
Pneumo 300 Doppler	0	0	2	4	2	3
Pneumo 500 Doppler	0	0	1	4	1	2
Pneumo 1200 Doppler	0	1	1	6	3	3

HEMO/PNEUMO SERIES ONLY	Left apex	Right apex	Left Mid	Right Mid	Left Base	Right Base
Baseline Doppler	0	0	1	1	2	1
Pneumo 150 Doppler	0	0	1	1	0	0
Pneumo 300 Doppler	0	0	1	4	2	1
Pneumo 500 Doppler	0	0	1	4	2	1
Hemo 250 Doppler	0	0	3	1	2	0
Hemo 250/Pneumo 250 Doppler	0	0	4	1	2	0

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